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                                                       ENTRY    SESSION
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=> s phytanic acid or phytenic acid or phytol or 14721-66-5/rn or 3653-46-1/rn or
150-86-7/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L3        5975 PHYTANIC ACID OR PHYTENIC ACID OR PHYTOL OR 14721-66-5/RN OR
               3653-46-1/RN OR 150-86-7/RN

=> s l3 and (phytanic or phytenic)
L4        2097 L3 AND (PHYTANIC OR PHYTENIC)

=> s l3 or (phytanic or phytenic)
L5        6013 L3 OR (PHYTANIC OR PHYTENIC)

=> s l5 and (diabetes or niddm or hyperinsulinemia or (insulin adj resistance))
L6          52 L5 AND (DIABETES OR NIDDM OR HYPERINSULINEMIA OR (INSULIN ADJ
               RESISTANCE))

=> dup rem l6
PROCESSING COMPLETED FOR L6
L7          36 DUP REM L6 (16 DUPLICATES REMOVED)

=> focus
PROCESSING COMPLETED FOR L7
L8          36 FOCUS L7 1-

=> d ibib abs hitstr 1-36

L8   ANSWER 1 OF 36  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:        2002:104617  CAPLUS
DOCUMENT NUMBER:         136:145248
TITLE:                   Use of phytanic acid for the
                         treatment of diabetes and other conditions
                         associated with impaired glucose tolerance
INVENTOR(S):             Fluehmann, Beat; Heim, Manuel; Hunziker, Willi; Weber,
                         Peter
PATENT ASSIGNEE(S):      Roche Vitamins A.-G., Switz.
SOURCE:                  Eur. Pat. Appl., 29 pp.
                         CODEN: EPXXDW
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:
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PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1177789	A2	20020206	EP 2001-118230	20010730
EP 1177789	A3	20030129		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 2002082298	A1	20020627	US 2001-915152	20010725
US 6784207	B2	20040831		
JP 2002104964	A2	20020410	JP 2001-233070	20010801
CA 2353805	AA	20020204	CA 2001-2353805	20010803
BR 2001003209	A	20020326	BR 2001-3209	20010803
CN 1365667	A	20020828	CN 2001-124878	20010803
US 2004138181	A1	20040715	US 2004-766118	20040127
PRIORITY APPLN. INFO.:			EP 2000-116848	A 20000804
			US 2001-915152	A3 20010725

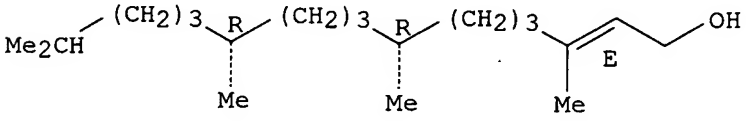
AB A method is provided for the treatment or prevention of preferably non-insulin dependent (**NIDDM** or so-called Type II) **diabetes** mellitus, or other conditions associated with impaired glucose tolerance, e.g. obesity, and in particular to the use of **phytanic acid** derivs. for the treatment or prevention.

IT **150-86-7, Phytol**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**phytanic acid** for the treatment of **diabetes** and other conditions associated with impaired glucose tolerance)

RN 150-86-7 CAPLUS

CN 2-Hexadecen-1-ol, 3,7,11,15-tetramethyl-, (2E,7R,11R)- (9CI) (CA INDEX NAME)

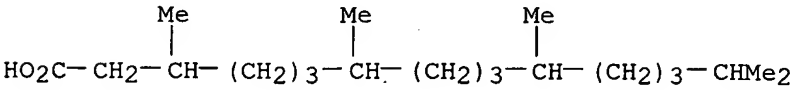
Absolute stereochemistry.
Double bond geometry as shown.



IT **14721-66-5, Phytanic acid**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**phytanic acid** for the treatment of **diabetes** and other conditions associated with impaired glucose tolerance)

RN 14721-66-5 CAPLUS

CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



L8 ANSWER 2 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:441439 CAPLUS

DOCUMENT NUMBER: 142:487490

TITLE: Use of **phytanic acid** for treating **diabetes**

INVENTOR(S): Zhou, Dingcheng

PATENT ASSIGNEE(S): Beiyi Medicine Sci. & Tech. Co., Ltd., Shanghai, Peop. Rep. China

SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, No pp.

given
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

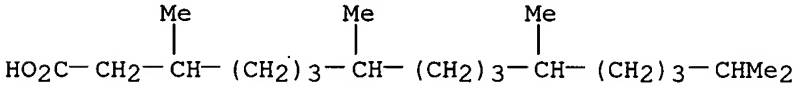
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1507871	A	20040630	CN 2002-154971	20021216

PRIORITY APPLN. INFO.: CN 2002-154971 20021216

AB The present invention relates to an application of **phytanic acid** for curing **diabetes**. Said invented medicine is prepared by adopting **phytanic acid** or its derivative and pharmaceutically-acceptable additive and/or adjuvant. The described derivative includes salts with alkali metal and alkali earth metal or their pharmaceutically-acceptable solvent compound The tests show that said invented medicine has good therapeutic effect for diabetic, specially, for patient with hyperglycemia, hyperlipemia and hypertension.

IT **14721-66-5, Phytanic acid**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(use of **phytanic acid** and its salts for treating **diabetes**)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



L8 ANSWER 3 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:343937 CAPLUS
DOCUMENT NUMBER: 137:304593
TITLE: **Phytanic acid**, a natural peroxisome proliferator-activated receptor agonist, regulates glucose metabolism in rat primary hepatocytes
AUTHOR(S): Heim, Manuel; Johnson, James; Boess, Franziska; Bendik, Igor; Weber, Peter; Hunziker, Willi; Fluehmann, Beat
CORPORATE SOURCE: Research and Development, Department of Human Nutrition and Health, Roche Vitamins, Basel, 4070, Switz.
SOURCE: FASEB Journal (2002), 16(7), 718-720, 10.1096/fj.01-0816fje
CODEN: FAJOEC; ISSN: 0892-6638
PUBLISHER: Federation of American Societies for Experimental Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

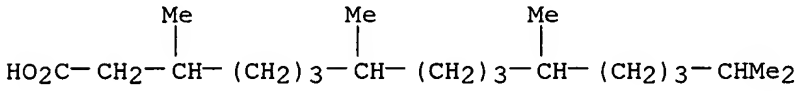
AB **Phytanic acid**, a metabolite of chlorophyll, is part of the human diet and is present in normal human serum at low micromolar concns. It was previously shown to be a ligand of the 9-cis-retinoic acid receptor and peroxisome proliferator-activated receptor (PPAR) α. PPAR agonists are widely used in the treatment of type 2 **diabetes**. This work reports that **phytanic acid** is not only a transactivator of PPARα, but it also acts via PPARβ and PPARγ in CV-1 cells cotransfected with the resp. full-length receptor and an acyl-CoA oxidase-PPAR-responsive element-luciferase construct. In contrast to other fatty acids, **phytanic**

acid at physiol. concns. enhanced the uptake of 2-deoxy-D-glucose in rat primary hepatocytes. This result could be explained by the increase in the expression of mRNAs for glucose transporters-1 and -2 and glucokinase, as determined by quant. real-time reverse transcriptase-polymerase chain reaction. Compared with the PPARγ-specific agonist ciglitazone, **phytanic acid** exerted only minor effects on the differentiation of C3H1OT1/2 cells into mature adipocytes. These results demonstrate that **phytanic acid** acts via different PPAR isoforms to modulate expression of genes involved in glucose metabolism, thus suggesting a potential role of **phytanic acid** in the management of insulin resistance.

IT **14721-66-5, Phytanic acid**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**phytanic acid**, a peroxisome proliferator-activated receptor agonist, regulation of glucose metabolism in hepatocytes)

RN 14721-66-5 CAPLUS

CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 4 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2001:219080 CAPLUS

DOCUMENT NUMBER: 135:175058

TITLE: The chlorophyll metabolite **phytanic acid** is a natural rexinoid - potential for treatment and prevention of **diabetes**

AUTHOR(S): McCarty, M. F.

CORPORATE SOURCE: Pantox Laboratories, San Diego, CA, 92109, USA

SOURCE: Medical Hypotheses (2001), 56(2), 217-219
CODEN: MEHYDY; ISSN: 0306-9877

PUBLISHER: Churchill Livingstone

DOCUMENT TYPE: Journal

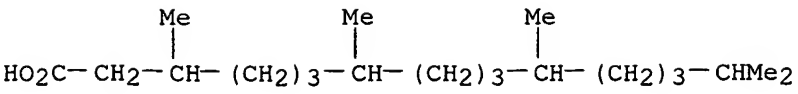
LANGUAGE: English

AB Synthetic ligands of the retinoid X receptor (RXR) have shown antidiabetic activity in mice, apparently owing to the fact that they stimulate the transcriptional activity of PPAR-γ/RXR heterodimers, much like thiazolidinedione drugs. The chlorophyll metabolite **phytanic acid** was shown to be a natural ligand for RXR, active in concns. near its physiol. levels. It is thus reasonable to suspect that **phytanic acid** may have utility for treatment and prevention of human type 2 **diabetes**. **Phytanic acid** may mimic or complement various effects of conjugated linoleic acids, which were shown to activate PPAR-γ/RXR and prevent rodent **diabetes**. Administration of hydrolyzed chlorophyll may represent the most cost-effective strategy for raising human tissue levels of **phytanic acid**.

IT **14721-66-5, Phytanic acid**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**phytanic acid** for treatment and prevention of **diabetes**)

RN 14721-66-5 CAPLUS

CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



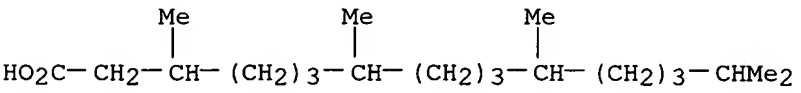
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 5 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:441299 CAPLUS
DOCUMENT NUMBER: 143:1297
TITLE: Medicine for treating **diabetes** and its complication
INVENTOR(S): Zhou, Dingcheng
PATENT ASSIGNEE(S): Shanghai Beiyi Medicine Sci-Tech Co., Ltd., Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, No pp. given
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1506049	A	20040623	CN 2002-150936	20021205
PRIORITY APPLN. INFO.:			CN 2002-150936	20021205

AB The medicine for treating **diabetes** and its complication is compounded with **phytanic acid** or its derivative, pharmaceutically acceptable additive and/or assistant. The derivative may be alkali metal salt or alkali earth metal salts of **phytanic acid** or their pharmaceutically acceptable solution Experiment shows that **phytanic acid** and its derivative have the activity of raising the taking of liver glucose and eliminating serum glucose and the activity is expressed by gene in enzyme inducing or stimulating insulin secretion. The present invention has excellent curative effect on **diabetes**, especially **diabetes** companied with hyperlipidemia, hypercholesterolemia, hypertension, obesity and **hyperinsulinemia**.

IT **14721-66-5, Phytanic acid**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(medicine for treating **diabetes** and its complication)
RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



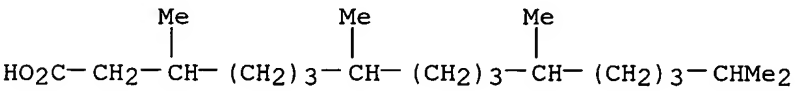
L8 ANSWER 6 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:624408 CAPLUS
DOCUMENT NUMBER: 138:348534
TITLE: The chlorophyll-derived metabolite **phytanic acid** induces white adipocyte differentiation
AUTHOR(S): Schlueter, A.; Yubero, P.; Iglesias, R.; Giralt, M.; Villarroya, F.
CORPORATE SOURCE: Department de Bioquimica i Biologia Molecular,

Universitat de Barcelona, Barcelona, Spain
SOURCE: International Journal of Obesity (2002), 26(9), 1277-1280
CODEN: IJOBDP; ISSN: 0307-0565
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Phytanic acid** is a derivative of the **phytol** side-chain of chlorophyll. It appears in humans following the ingestion of fat-containing foods and is present in human blood at a low micromolar concentration It may activate retinoid X receptors (RXR) or peroxisome proliferator-activated receptor (PPAR) α in vitro. **Phytanic acid** induced the adipocyte differentiation of 3T3-L1 cells in culture as assessed by accumulation of lipid droplets and induction of the aP2 mRNA marker. This effect was mimicked by a synthetic activator of RXR but not by a PPARα agonist or by palmitic acid. In human pre-adipocytes in primary culture, **phytanic acid** also induced adipocyte differentiation. These findings indicate that **phytanic acid** may act as a natural rexinoid in adipose cells and suggest a potential use in the treatment of human type 2 **diabetes** and obesity.

IT **14721-66-5, Phytanic acid**
RL: DMA (Drug mechanism of action); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chlorophyll-derived metabolite **phytanic acid** induces white adipocyte differentiation)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 7 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:949902 CAPLUS
DOCUMENT NUMBER: 142:328541
TITLE: Nutraceutical resources for **diabetes** prevention - an update
AUTHOR(S): McCarty, Mark F.
CORPORATE SOURCE: NutriGuard Research, Encinitas, CA, 92024, USA
SOURCE: Medical Hypotheses (2004), Volume Date 2005, 64(1), 151-158
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. There is considerable need for safe agents that can reduce risk for **diabetes** in at-risk subjects. Although certain drugs - including metformin, acarbose, and orlistat - have shown **diabetes** -preventive activity in large randomized studies, nutraceuticals have potential in this regard as well. Natural agents which slow carbohydrate absorption may mimic the protective effect of acarbose; these include: soluble fiber - most notably glucomannan; chlorogenic acid - likely responsible for reduction in **diabetes** risk associated with heavy coffee intake; and legume-derived α-amylase inhibitors. There does not appear to be a natural lipase inhibitor functionally equivalent to orlistat, although there are poorly documented claims for Cassia nomame exts. Metformin's efficacy reflects activation of AMP-activated kinase; there is

preliminary evidence that certain compds. in barley malt have similar activity, without the side effects associated with metformin. In supraphysiol. concns., biotin directly activates soluble guanylate cyclase; this implies that, at some sufficient intake, biotin should exert effects on β cells, the liver, and skeletal muscle that favor good glucose tolerance and maintenance of effective β cell function. Good magnesium status is associated with reduced **diabetes** risk and superior insulin sensitivity in recent epidemiol.; ample intakes of chromium picolinate appear to promote insulin sensitivity in many individuals and improve glycemic control in some diabetics; calcium/vitamin D may help preserve insulin sensitivity by preventing secondary hyperparathyroidism. Although conjugated linoleic acid - like thiazolidinediones, a PPAR-γ agonist - has not aided insulin sensitivity in clin. trials, the natural rexinoid **phytanic acid** exerts thiazolidinedione-like effect in animals and cell cultures, and merits clin. examination Other natural agents with the potential to treat and possibly prevent **diabetes** include exts. of bitter melon and of cinnamon. Nutraceuticals featuring meaningful doses of combinations of these agents would likely have substantial **diabetes**-preventive efficacy, and presumably could be marketed legally as aids to good glucose tolerance and insulin sensitivity.

REFERENCE COUNT: 123 THERE ARE 123 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 8 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:182642 CAPLUS
DOCUMENT NUMBER: 140:216524
TITLE: Novel nutraceutical compositions comprising biotin
INVENTOR(S): Eggersdorfer, Manfred Ludwig; Raederstorff, Daniel; Teixeira, Sandra Renata; Weber, Peter
PATENT ASSIGNEE(S): DSM IP Assets B.V., Neth.
SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004017766	A1	20040304	WO 2003-EP9112	20030818
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003266287	A1	20040311	AU 2003-266287	20030818
EP 1536698	A1	20050608	EP 2003-792352	20030818
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1678203	A	20051005	CN 2003-819982	20030818
JP 2005536543	T2	20051202	JP 2004-530180	20030818
US 2005256178	A1	20051117	US 2005-525348	20050222

PRIORITY APPLN. INFO.:

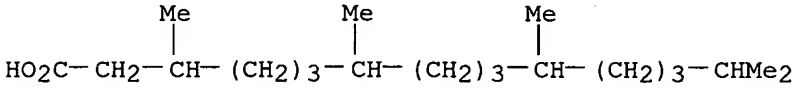
EP 2002-18847	A	20020823
EP 2003-14625	A	20030626
WO 2003-EP9112	W	20030818

AB Nutraceutical compns. comprise biotin in an amount sufficient to administer

to a subject a daily dosage of 0.01 mg per kg body weight to about 3 mg per kg body weight and at least one addnl. component selected from pantethine or a metabolite thereof, EGCG, **phytanic acid**, lipoic acid and policosanol. The compns. are useful for the treatment of both type 1 and 2 **diabetes**, and for the prevention of type 2 **diabetes** in those individuals with pre-**diabetes**, or impaired glucose tolerance (IGT) or obesity.

IT **14721-66-5, Phytanic acid**
RL: FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nutraceutical compns. comprising biotin for treatment of **diabetes**, glucose tolerance and obesity)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 10 THERE ARE 10 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 9 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:283266 CAPLUS
DOCUMENT NUMBER: 142:309913
TITLE: Compositions for the treatment and prevention of **diabetes** mellitus
INVENTOR(S): Raederstorff, Daniel; Teixeira, Sandra Renata; Wang, Ying; Weber, Peter; Wolfram, Swen
PATENT ASSIGNEE(S): DSM IP Assets B.V., Neth.
SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

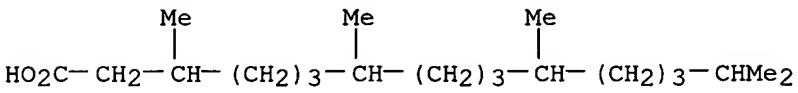
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005027661	A1	20050331	WO 2004-EP10283	20040915
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
EP 1662906	A1	20060607	EP 2004-765197	20040915
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				

PRIORITY APPLN. INFO.: EP 2003-21447 A 20030923
WO 2004-EP10283 W 20040915

AB Compns. comprising a catechin as found in green tea, e.g., epigallocatechin gallate, and ligand which activates the peroxisome proliferator-activated receptor gamma (PPAR-gamma are useful for the treatment and prevention of **diabetes** mellitus).

IT **14721-66-5, Phytanic acid**

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compns. for treatment and prevention of **diabetes** mellitus)
RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 10 OF 36 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:396833 BIOSIS
DOCUMENT NUMBER: PREV200400402240
TITLE: **Phytanic acid** derivative compositions.
AUTHOR(S): Fluehmann, Beat [Inventor, Reprint Author]; Hunziker, Willi [Inventor]
CORPORATE SOURCE: Zurich, Switzerland
ASSIGNEE: Roche Vitamins Inc.
PATENT INFORMATION: US 6784207 20040831
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Aug 31 2004) Vol. 1285, No. 5. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Oct 2004
Last Updated on STN: 13 Oct 2004
AB The present invention is a method for the treatment or prevention of preferably non-insulin dependent (**NIDDM** or so-called Type II) **diabetes** mellitus, or other conditions associated with impaired glucose tolerance such as obesity, and in particular to the use of **phytanic acid** derivatives for the said treatment and/or prevention. A method of making a composition for the treatment or prevention of non-insulin dependent **diabetes** mellitus and related diseases comprising combining **phytanic acid** or derivatives thereof with a pharmaceutically acceptable additive or adjuvant, and a composition for the treatment or prevention of non-insulin dependent **diabetes** mellitus comprising **phytanic acid** or derivatives thereof are also provided.

L8 ANSWER 11 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1220665 CAPLUS
DOCUMENT NUMBER: 143:466228
TITLE: Use of targeted oxidative therapeutic formulation in treatment of **diabetes** and obesity
INVENTOR(S): Hofmann, Robert F.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 34 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005107728	A2	20051117	WO 2005-US15846	20050505

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2005272714 A1 20051208 US 2005-122907 20050505

PRIORITY APPLN. INFO.: US 2004-568542P P 20040506

AB A pharmaceutical formulation contains peroxide species or reaction products resulting from oxidation of an alkene, such as geraniol, by an oxidizing agent, such as ozone; a penetrating solvent, such as DMSO; a dye containing a chelated metal, such as hematoporphyrin; and an aromatic redox compound, such as benzoquinone. The formulation is used to effectively treat patients affected with **diabetes** and obesity. Thus, geraniol was subjected to ozonolysis, and the ozonized product 0.54, DMSO 98.00, hematoporphyrin 0.83, methylnaphthoquinone 0.24, and Rose bengal 0.39%.

L8 ANSWER 12 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2004:251322 CAPLUS

DOCUMENT NUMBER: 140:385310

TITLE: Retinoids and retinoid receptors in the control of energy balance: novel pharmacological strategies in obesity and **diabetes**

AUTHOR(S): Villarroya, F.; Iglesias, R.; Giralt, M.

CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Barcelona, Barcelona, E-08028, Spain

SOURCE: Current Medicinal Chemistry (2004), 11(6), 795-805
CODEN: CMCHE7; ISSN: 0929-8673

PUBLISHER: Bentham Science Publishers Ltd.

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review. Obesity and type II **diabetes** are closely related metabolic diseases with an increasing incidence worldwide. No clear-cut pharmacol. treatment for these complex metabolic disturbances is available despite current efforts. New directions and perspectives for the pharmacol. or nutritional treatment of these diseases should be defined. In recent years, a growing body of evidence shows that retinoids and retinoic acid receptors are involved in the control of biol. aspects (e.g. adiposity and energy expenditure mechanisms), which offers great potential for research on the treatment of obesity and type II **diabetes**. All-trans retinoic acid is known to inhibit adipocyte differentiation, whereas, mols. activating the retinoid X-receptor (rexinoids) promote the differentiation of adipocytes. Treatment with rexinoids ameliorates glycemic control in rodent models of type II **diabetes** and obesity, although other findings indicate similar pos. effects by inhibiting the receptor. Moreover, natural products of dietary origin, such as **phytanic acid** can activate RXR and thus, trigger adipose cell differentiation. Finally, the activation of retinoic acid receptors or retinoid X receptors has been reported to induce the gene expression of uncoupling proteins, which are mitochondrial proteins involved in the regulation of energy expenditure and fatty acid metabolism Further research is required to exploit the capacities of the retinoid-dependent pathways of regulation of adiposity, insulin sensitivity and energy expenditure for drug development in metabolic disturbances.

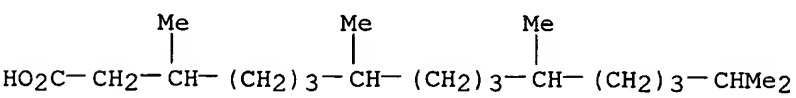
REFERENCE COUNT: 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR

THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 13 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:1088101 CAPLUS
DOCUMENT NUMBER: 143:4824
TITLE: Up-regulation of PPARγ coactivator-1α as a strategy for preventing and reversing insulin resistance and obesity
AUTHOR(S): McCarty, Mark F.
CORPORATE SOURCE: NutriGuard Research, Encinitas, CA, 92024, USA
SOURCE: Medical Hypotheses (2005), 64(2), 399-407
CODEN: MEHYDY; ISSN: 0306-9877
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Excessive accumulation of triglycerides and certain fatty acid derivs. in skeletal muscle and other tissues appears to mediate many of the adverse effects of insulin resistance syndrome. Although fatty diets and obesity can promote such accumulation, deficient capacity for fatty acid oxidation can also contribute in this regard. Indeed, in subjects who are insulin resistant, diabetic, and/or obese, fatty acid oxidation by skeletal muscle tends to be inefficient, reflecting decreased expression of mitochondria and mitochondrial enzymes in muscle. This phenomenon is not corrected by weight loss, is not simply reflective of subnormal phys. activity, and is also seen in lean first-degree relatives of diabetics; thus, it appears to be primarily attributable to genetic factors. Recent studies indicate that decreased expression of PPARγ coactivator-1α (PGC-1α), a "master switch" which induces mitochondrial biogenesis by supporting the transcriptional activity of the nuclear respiratory factors, may largely account for the diminished oxidative capacity of subjects prone to insulin resistance. Thus, feasible measures which up-regulate PGC-1α may be useful for preventing and treating insulin resistance and obesity. These may include exercise training, metformin and other agents which stimulate AMP-activated kinase, high-dose biotin, and PPARδ agonists. Drugs which are specific agonists for PPARδ show remarkable efficacy in rodent models of insulin resistance, **diabetes**, and obesity, and are currently being evaluated clin. **Phytanic acid**, a branched-chain fatty acid found in omnivore diets, can also activate PPARδ, and thus should be examined with respect to its impact on mitochondrial biogenesis and insulin sensitivity.

IT **14721-66-5, Phytanic acid**
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(phytaic acid branch chain fatty acid found in omnivore diets can also activate PPARα)
RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 114 THERE ARE 114 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

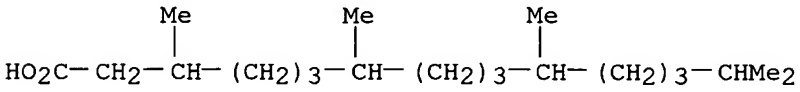
L8 ANSWER 14 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1972:473445 CAPLUS
DOCUMENT NUMBER: 77:73445

TITLE: Plasma free fatty acids and obesity
AUTHOR(S): Badinand, A.; Losman, M.
CORPORATE SOURCE: Lab. Cent. Chim. Biol., Hop. E. Herriot, Lyons, Fr.
SOURCE: Bollettino Chimico Farmaceutico (1972), 111(3), 147-58
CODEN: BCFAAI; ISSN: 0006-6648
DOCUMENT TYPE: Journal
LANGUAGE: Italian

AB Anal. of plasma free fatty acids and adipose tissue fatty acids of 8 human controls, 18 obese subjects, and 5 diabetics by thin-layer and gas chromatog. showed a higher concentration of stearic and palmitic acid in the plasma than in adipose tissue, particularly in obese subjects. In contrast, concentration of oleic acid is higher in adipose tissue. Its concentration is lowest in some obese subjects. The relatively high concentration of **phytanic acid** in plasma in comparison to adipose tissue indicate that its origin is not endogenous.

IT **14721-66-5**
RL: BIOL (Biological study)
(of blood plasma, in obesity, **diabetes** in relation to)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



L8 ANSWER 15 OF 36 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2003103483 EMBASE
TITLE: **Phytanic acid** alpha-oxidation, new insights into an old problem: A review.
AUTHOR: Wanders R.J.A.; Jansen G.A.; Lloyd M.D.
CORPORATE SOURCE: R.J.A. Wanders, Depts. Pediat./Emma Children's H., Academic Medical Centre, University Hospital Amsterdam, P.O. Box 22700, 1100 DE Amsterdam, Netherlands. r.j.wanders@amc.uva.nl
SOURCE: Biochimica et Biophysica Acta - Molecular and Cell Biology of Lipids, (17 Mar 2003) Vol. 1631, No. 2, pp. 119-135. .
Refs: 91
ISSN: 1388-1981 CODEN: BBMLFG
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 25 Mar 2003
Last Updated on STN: 25 Mar 2003

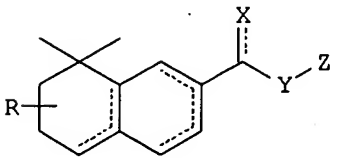
AB **Phytanic acid** (3,7,10,14-tetramethylhexadecanoic acid) is a branched-chain fatty acid which is known to accumulate in a number of different genetic diseases including Refsum disease. Due to the presence of a methyl-group at the 3-position, **phytanic acid** and other 3-methyl fatty acids can not undergo β-oxidation but are first subjected to fatty acid α-oxidation in which the terminal carboxyl-group is released as CO(2). The mechanism of α-oxidation has long remained obscure but has been resolved in recent years. Furthermore, peroxisomes have been found to play an indispensable role in fatty acid α-oxidation, and the complete α-oxidation machinery is probably localized in peroxisomes. This Review describes the current state of knowledge about fatty acid α-oxidation in mammals with

particular emphasis on the mechanism involved and the enzymology of the pathway. .COPYRGT. 2003 Elsevier Science B.V. All rights reserved.

L8 ANSWER 16 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:368900 CAPLUS
DOCUMENT NUMBER: 140:395235
TITLE: Nuclear hormone receptor compounds such as β-ionol and fatty acid analogs for the treatment of cancer and skin disorders.
INVENTOR(S): Delong, Mitchell Anthony; Biedermann, Kimberly Ann; Bissett, Donald Lynn; Boyer, Angelique Sun; Cohen, Scott Louis; Snider, Catherine Elizabeth
PATENT ASSIGNEE(S): The Procter & Gamble Company, USA
SOURCE: PCT Int. Appl., 83 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004037213	A2	20040506	WO 2003-US34155	20031023
WO 2004037213	A3	20040729		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004131648	A1	20040708	US 2002-279397	20021024
CA 2500974	AA	20040506	CA 2003-2500974	20031023
AU 2003285042	A1	20040513	AU 2003-285042	20031023
EP 1553916	A2	20050720	EP 2003-779359	20031023
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
CN 1705468	A	20051207	CN 2003-80101584	20031023
JP 2006507287	T2	20060302	JP 2004-547235	20031023
PRIORITY APPLN. INFO.:			US 2002-279397 A	20021024
			WO 2003-US34155 W	20031023

OTHER SOURCE(S): MARPAT 140:395235
GI



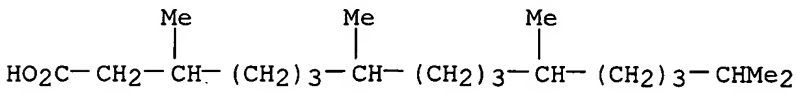
I

AB Title compds. e.g. [I; X = single or double bonded moiety comprising 0-12 (substituted) C atoms, 0-2 heteroatoms; Z = single, double, or triple bonded moiety comprising 0-12 C atoms in a chain, optionally including (substituted) cycloalkyl, aryl rings; Y = (CH2)n; n = 0-3; R =

(substituted) alkyl, cycloalkyl, aryl], are claimed. (no synthetic data). Title compds. are believed to function as RXR, RAR and/or PPAR receptor ligands to encourage skin differentiation and discourage excess skin proliferation.

IT **14721-66-5, Phytanic acid**
RL: COS (Cosmetic use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nuclear hormone receptor compds. such as β-ionol and fatty acid analogs for the treatment of cancer and skin disorders)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



L8 ANSWER 17 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1963:485146 CAPLUS
DOCUMENT NUMBER: 59:85146
ORIGINAL REFERENCE NO.: 59:15823e-g
TITLE: The effect of N2-butylbiguanide (W 37) and N1-(β-phenylethyl)biguanide (W 32) upon alioxan- and phlorizin-induced **diabetes** and the intestinal glucose absorption in rats
AUTHOR(S): Creutzfeldt, W.; Soeling, H. D.; Moench, A.; Rauh, E.; Bol, M.
CORPORATE SOURCE: Med. Univ.-Klin., Frieberg i. Br., Germany
SOURCE: Archiv fuer Experimentelle Pathologie und Pharmakologie (1962), 244(1), 31-47
CODEN: AEXPBL; ISSN: 0365-2041
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB Rats with alloxaninduced **diabetes** of over 4-months duration showed no noticeable blood sugar decrease for 4-8 hrs. after subcutaneous injection of either 80 mg. W-37/kg. or of 70 mg. W-32/kg. Three injections of 20-30 mg. W-37/kg. decreased glycosuria. This symptom is due not to an increase in glucose utilization, but to a change in kidney function. In rats, rendered diabetic by 3 daily doses of 250 mg. phlorizin/kg., W-37 caused no decrease in glycosuria. The effect of W-37 on glucose absorption was tested by addition of 50-100 mg./kg. to glucose-filled small intestine, tied off in situ. No decrease in glucose absorption took place even after preliminary rinsing of the intestines with W-37. Administered subcutaneously, the same dose caused slight, temporary decrease in glucose absorption after 100-200 min. Stomach emptying time was prolonged by W-37. 26 references.

L8 ANSWER 18 OF 36 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:288048 BIOSIS
DOCUMENT NUMBER: PREV200400286805
TITLE: Induction of Inflammatory Markers in Epididymal Adipose Tissue of Diet-Induced Obese (DIO) C57BL/6J Mice: Impact of **Phytanic Acid** and BRL49653.
AUTHOR(S): Teixeira, Sandra R [Reprint Author]; Preller, Mareike; Wang, Ying; Schwager, Joseph; Champy, Marie-France; Auwerx, Johan; Elste, Volker; Weber, Peter; Fluehmann, Beat
CORPORATE SOURCE: R&&AMPD Human Nutrition and Health, DSM Nutritional Products, P.O: Box 3255, Bldg 205/209B, Basel, 4002, Switzerland
sandra-renata.teixeira@dsm.com

SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 356.13. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

AB The innate immune system and the stimulation of acute-phase protein synthesis in liver have been postulated to contribute to insulin resistance and T2DM. In this study, we examined the effect of diet-induced obesity on gene expression of inflammatory markers in adipose tissue. 48 male C57BL/6J mice were assigned to 4 groups (n=12/group). One group received chow (lean control, LC), while 3 groups received a high-fat (HF) diet. One of the HF groups served as the fat control (FC), whereas the other 2 received additionally either **phytanic acid** at 150 mpk or BRL49653 at 10 mpk (TZD). Mice receiving HF became obese and diabetic during the study period. After 23wks, epididymal adipose tissue was collected from 6 mice/group and analyzed using Affymetrix Genechip. Genes known to be involved in inflammatory responses were selected and further filtered to include only those with change factors <-0.5 or >0.5 and p-value <0.05. HF diet resulted in upregulation of the acute-phase proteins haptoglobin, and orosomucoid 1 and 2, the lipopolysaccharide (LPS) binding protein, and heat-shock protein (HSP) 72. Treatment with either PPARgamma agonist resulted in a downregulation of the expression of most of these markers to levels close to LC. Other classical inflammatory markers were not regulated. Our results with selected inflammatory markers suggest that diet-induced obesity induces a persistent acute-phase reaction in adipose tissue, which may contribute to insulin-resistance. Moreover, the two investigated PPARgamma agonists can reduce the amount of inflammation, while improving metabolic status.

L8 ANSWER 19 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:412796 CAPLUS
DOCUMENT NUMBER: 140:395555
TITLE: Antidiabetic nutraceutical compositions comprising epigallocatechin gallate
INVENTOR(S): Raederstorff, Daniel; Teixeira, Sandra Renata; Weber, Peter
PATENT ASSIGNEE(S): DSM Ip Assets B.V., Neth.
SOURCE: PCT Int. Appl., 21 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004041257	A2	20040521	WO 2003-EP10838	20030930
WO 2004041257	A3	20040805		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR,

BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

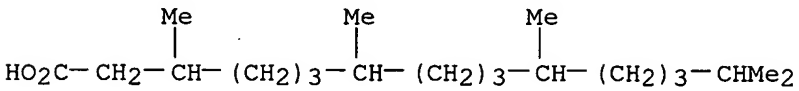
AU 2003293592	A1	20040607	AU 2003-293592		20030930
EP 1558244	A2	20050803	EP 2003-788928		20030930
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK					
CN 1694695	A	20051109	CN 2003-824662		20030930
JP 2006508096	T2	20060309	JP 2004-548728		20030930
US 2006165671	A1	20060727	US 2005-533858		20051212
PRIORITY APPLN. INFO.:			EP 2002-24804	A	20021107
			WO 2003-EP10838	W	20030930

AB The invention relates to nutraceutical compns. comprising at least two ingredients from the groups of epigallocatechin gallate, pantethine or a metabolite thereof, **phytanic acid**, lipoic acid, policosanol and coenzyme Q-10 and their use in the treatment or prevention of **diabetes** or obesity.

IT **14721-66-5, Phytanic acid**
RL: FFD (Food or feed use); MOA (Modifier or additive use); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(antidiabetic nutraceutical compns. comprising epigallocatechin gallate)

RN 14721-66-5 CAPLUS

CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



L8 ANSWER 20 OF 36 MEDLINE on STN

ACCESSION NUMBER: 2004562288 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15533633
TITLE: Nutraceutical resources for **diabetes** prevention--an update.
AUTHOR: McCarty Mark F
CORPORATE SOURCE: NutriGuard Research, 1051 Hermes Avenue, Encinitas, CA 92024, USA.. mccarty@pantox.com
SOURCE: Medical hypotheses, (2005) Vol. 64, No. 1, pp. 151-8. Journal code: 7505668. ISSN: 0306-9877.
PUB. COUNTRY: Scotland: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 10 Nov 2004
Last Updated on STN: 22 Apr 2005
Entered Medline: 21 Apr 2005

AB There is considerable need for safe agents that can reduce risk for **diabetes** in at-risk subjects. Although certain drugs--including metformin, acarbose, and orlistat--have shown **diabetes** -preventive activity in large randomized studies, nutraceuticals have potential in this regard as well. Natural agents which slow carbohydrate absorption may mimic the protective effect of acarbose; these include: soluble fiber--most notably glucomannan; chlorogenic acid--likely responsible for reduction in **diabetes** risk associated with heavy coffee intake; and legume-derived alpha-amylase inhibitors. There does not appear to be a natural lipase inhibitor functionally equivalent to orlistat, although there are poorly documented claims for Cassia nomame extracts. Metformin's efficacy reflects activation of AMP-activated kinase; there is preliminary evidence that certain compounds in barley malt have similar activity, without the side effects associated with metformin. In supraphysiological concentrations, biotin directly

activates soluble guanylate cyclase; this implies that, at some sufficient intake, biotin should exert effects on beta cells, the liver, and skeletal muscle that favor good glucose tolerance and maintenance of effective beta cell function. Good magnesium status is associated with reduced **diabetes** risk and superior insulin sensitivity in recent epidemiology; ample intakes of chromium picolinate appear to promote insulin sensitivity in many individuals and improve glycemic control in some diabetics; calcium/vitamin D may help preserve insulin sensitivity by preventing secondary hyperparathyroidism. Although conjugated linoleic acid--like thiazolidinediones, a PPAR-gamma agonist--has not aided insulin sensitivity in clinical trials, the natural rexinoid **phytanic acid** exerts thiazolidinedione-like effect in animals and cell cultures, and merits clinical examination. Other natural agents with the potential to treat and possibly prevent **diabetes** include extracts of bitter melon and of cinnamon. Nutraceuticals featuring meaningful doses of combinations of these agents would likely have substantial **diabetes**-preventive efficacy, and presumably could be marketed legally as aids to good glucose tolerance and insulin sensitivity.

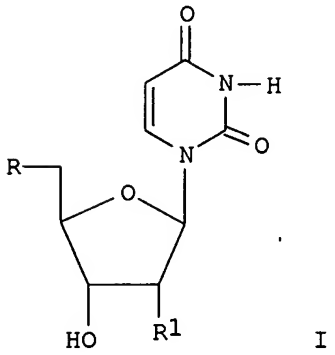
L8 ANSWER 21 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:849656 CAPLUS
DOCUMENT NUMBER: 137:338098
TITLE: Preparation of pharmaceutically active uridine ester nucleosides against a variety of diseases
INVENTOR(S): Susilo, Rudy
PATENT ASSIGNEE(S): Germany
SOURCE: PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002088159	A1	20021107	WO 2002-EP4725	20020429
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2445861	AA	20021107	CA 2002-2445861	20020429
EE 200300536	A	20040216	EE 2003-536	20020429
EP 1390378	A1	20040225	EP 2002-766645	20020429
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
CN 1505636	A	20040616	CN 2002-809160	20020429
BR 2002009320	A	20040720	BR 2002-9320	20020429
JP 2004531543	T2	20041014	JP 2002-585457	20020429
NZ 528634	A	20050429	NZ 2002-528634	20020429
EP 1666092	A2	20060607	EP 2005-17150	20020429
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, MK, CY, AL, TR				
NO 2003004782	A	20031212	NO 2003-4782	20031024
US 2004121979	A1	20040624	US 2003-476287	20031029
BG 108299	A	20040930	BG 2003-108299	20031029
ZA 2003008420	A	20041029	ZA 2003-8420	20031029
US 2005043269	A1	20050224	US 2004-951764	20040929

US 2005043394	A1	20050224	US 2004-951776		20040929
US 2005065110	A1	20050324	US 2004-951724		20040929
AU 2006200874	A1	20060323	AU 2006-200874		20060301
PRIORITY APPLN. INFO.:			EP 2001-110608	A	20010430
			US 2001-288090P	P	20010503
			EP 2001-124879	A	20011018
			US 2001-330429P	P	20011022
			EP 2002-766645	A3	20020429
			WO 2002-EP4725	W	20020429
			US 2003-476287	A3	20031029

OTHER SOURCE(S): MARPAT 137:338098

GI



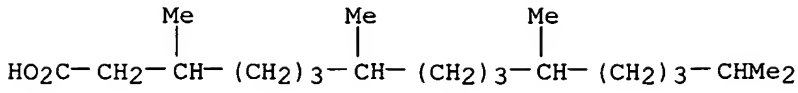
I

AB The present invention relates to novel uridine esters I, wherein R represents a carboxylic acid residue, preferably a fatty acid residue and R1 represents hydrogen or a hydroxy group, their use as pharmaceutically active agents against a variety of diseases, methods for the preparation of said uridine esters and pharmaceutical compns. containing at least one uridine ester as active ingredient. The present invention relates also to a drug combination comprising free fatty acids and/or fatty acid esters and uridine, deoxyuridine, uridine monophosphate and/or deoxyuridine monophosphate, and to the use of such a drug combination. Thus, I [R = OCO(CH:CHCH2)6Et, R1 = OH] was prepared and tested in NMRI mice against a variety of diseases such as **diabetes**, polyneuropathy, and neuroprotective effects. Title compds were prepared as stimulant drug and/or for prophylaxis and/or treatment of **diabetes** mellitus Type I and Type II, inflammation, cancer, necrosis, gastric ulcers, neurodegenerative diseases (Alzheimer's disease, Parkinson's disease), neuropathic diseases, neuropathic pain and polyneuropathy, peripheral and/or central nerve diseases, degradation of the peripheral and/or central nerve system, heavy metal poisoning, ischemic diseases and ischemic heart disease, liver diseases and dysfunction of liver, allergies, cardiovascular diseases, Chlamydia pneumoniae, depression, obesity, stroke, pain, and/or retroviral infections (HIV, AIDS), including opportunistic infections. Dihomo-γ-linolenic acid Arachidonic acid 7,10,13,16-Docosatetraenoic acid α-Linolenic acid Stearidonic acid 8,11,14,17-Eicosatetraenoic acid γ-Linolenic acid.

IT **14721-66-5, Phytanic acid**
RL: BSU (Biological study, unclassified); BIOL (Biological study) (preparation of)

RN 14721-66-5 CAPLUS

CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 22 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:795639 CAPLUS
DOCUMENT NUMBER: 145:195780
TITLE: Compositions comprising epigallocatechin gallate and protein hydrolysate
INVENTOR(S): Wolfram, Swen
PATENT ASSIGNEE(S): Dsm Ip Assets B.V., Neth.
SOURCE: PCT Int. Appl., 38pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2006082222	A1	20060810	WO 2006-EP50623	20060202

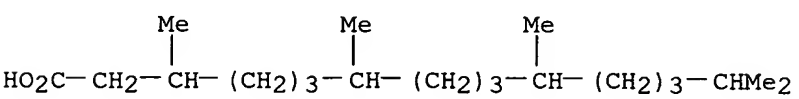
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: EP 2005-100755 A 20050203

AB The present invention describes a composition comprising EGCG and a protein hydrolyzate.

IT **14721-66-5, Phytanic acid**
RL: PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(compns. comprising epigallocatechin gallate and protein hydrolyzate)

RN 14721-66-5 CAPLUS
CN Hexadecanoic acid, 3,7,11,15-tetramethyl- (8CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L8 ANSWER 23 OF 36 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:737602 CAPLUS
DOCUMENT NUMBER: 139:244708
TITLE: Immunomodulatory polymeric antigens for treating inflammatory diseases
INVENTOR(S): Taylor, Kathleen Ann; Blaszczak, Larry Chris;

Blackburn, Neil Thomas
PATENT ASSIGNEE(S): Eli Lilly and Company, USA
SOURCE: PCT Int. Appl., 96 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

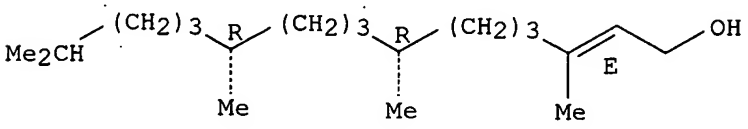
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003075953	A2	20030918	WO 2003-US5575	20030307
WO 2003075953	A3	20040401		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003217685	A1	20030922	AU 2003-217685	20030307
EP 1494687	A2	20050112	EP 2003-713643	20030307
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 2005119164	A1	20050602	US 2003-506312	20030307
PRIORITY APPLN. INFO.:			US 2002-363065P P	20020308
			US 2002-365211P P	20020315
			WO 2003-US5575 W	20030307

AB Provided are natural and synthetic immunomodulatory polymeric antigens (SPAs); compns. containing SPAs, Streptococcus pneumoniae capsule-derived CP1 and mixts.; as well as methods of using these natural and synthetic SPAs and compns. to prevent or treat inflammatory pathologies. A novel synthetic peptidoglycan was prepared for the purpose of the invention.

IT **150-86-7, Phytol**
RL: RCT (Reactant); RACT (Reactant or reagent)
(immunomodulatory polymeric antigens for treating inflammatory diseases)

RN 150-86-7 CAPLUS
CN 2-Hexadecen-1-ol, 3,7,11,15-tetramethyl-, (2E,7R,11R)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.



L8 ANSWER 24 OF 36 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2001263903 EMBASE
TITLE: PPARγ/RXR as a molecular target for **diabetes**
AUTHOR: Lenhard J.M.
CORPORATE SOURCE: J.M. Lenhard, Department of Metabolic Diseases, GlaxoSmithKline Inc., 5 Moore Drive, Research Triangle Park, NC 27709, United States

SOURCE: Receptors and Channels, (2001) Vol. 7, No. 4, pp. 249-258.
Refs: 141
ISSN: 1060-6823 CODEN: RCHAE4
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Aug 2001
Last Updated on STN: 16 Aug 2001

AB Type 2 **diabetes** is associated with insulin resistance in peripheral tissues, such as muscle and fat. Novel therapies that improve insulin action include ligands that bind and activate the nuclear receptors peroxisome proliferator activating receptor γ (PPARγ) and retinoid X receptor (RXR). PPARγ/RXR form heterodimers that regulate transcription of genes involved in insulin action, adipocyte differentiation, lipid metabolism and inflammation. PPARγ activators include prostanoids, fatty acids, thiazolidinediones and N-(2-benzoylphenyl)tyrosine analogues. RXR ligands include naturally occurring retinoic acid and synthetic rexinoids. Selective ligands for these receptors improve metabolic abnormalities associated with type 2 **diabetes**, such as hyperglycemia, hyperlipidemia, insulin resistance and other cardiovascular risk factors. Although adipose tissue mediates some of the effects of PPARγ/RXR ligands, other tissues also regulate the effects of these receptors. The activity of the PPARγ/RXR heterodimer is influenced by posttranslational modifications, receptor turnover, polymorphisms, splice variants, coactivators and corepressors. This article reviews recent developments in research on these receptors, with particular emphasis on metabolic effects, ligand selectivity, structure and regulation of the PPARγ/RXR heterodimer.

L8 ANSWER 25 OF 36 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:154697 BIOSIS
DOCUMENT NUMBER: PREV200600162521
TITLE: **Phytanic acid** improves metabolic parameters and modulates hepatic gene expression in vivo (DIO C57BL/6J mice).
AUTHOR(S): Preller, Mareike [Reprint Author]; Wang, Ying; Champy, Marie-France; Auwerx, Johan; Elste, Volker; Fluehmann, Beat; Weber, Peter; Teixeira, Sandra
SOURCE: Diabetes, (JUN 2004) Vol. 53, No. Suppl. 2, pp. A266. Meeting Info.: 64th Annual Meeting of the American-Diabetes-Association. Orlando, FL, USA. June 04 -08, 2004. Amer Diabet Assoc.
CODEN: DIAEAZ. ISSN: 0012-1797.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Mar 2006
Last Updated on STN: 9 Mar 2006

L8 ANSWER 26 OF 36 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2002164911 EMBASE
TITLE: The mode of action of thiazolidinediones.

AUTHOR: Hauner H.
CORPORATE SOURCE: H. Hauner, German Diabetes Research Institute, Heinrich-Heine University, Auf'm Hennekamp 65, D-40225 Dusseldorf, Germany. hauner@dfi.uni-duesseldorf.de
SOURCE: Diabetes/Metabolism Research and Reviews, (2002) Vol. 18, No. SUPPL. 2, pp. S10-S15. .
Refs: 59
ISSN: 1520-7552 CODEN: DMRRFM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
022 Human Genetics
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 May 2002
Last Updated on STN: 23 May 2002

AB The thiazolidinediones (TZDs) or 'glitazones' are a new class of oral antidiabetic drugs that improve metabolic control in patients with type 2 **diabetes** through the improvement of insulin sensitivity. TZDs exert their antidiabetic effects through a mechanism that involves activation of the gamma isoform of the peroxisome proliferator-activated receptor (PPARγ), a nuclear receptor. TZD-induced activation of PPARγ alters the transcription of several genes involved in glucose and lipid metabolism and energy balance, including those that code for lipoprotein lipase, fatty acid transporter protein, adipocyte fatty acid binding protein, fatty acyl-CoA synthase, malic enzyme, glucokinase and the GLUT4 glucose transporter. TZDs reduce insulin resistance in adipose tissue, muscle and the liver. However, PPARγ is predominantly expressed in adipose tissue. It is possible that the effect of TZDs on insulin resistance in muscle and liver is promoted via endocrine signalling from adipocytes. Potential signalling factors include free fatty acids (FFA) (well-known mediators of insulin resistance linked to obesity) or adipocyte-derived tumour necrosis factor-α (TNF-α), which is overexpressed in obesity and insulin resistance. Although there are still many unknowns about the mechanism of action of TZDs in type 2 **diabetes**, it is clear that these agents have the potential to benefit the full 'insulin resistance syndrome' associated with the disease. Therefore, TZDs may also have potential benefits on the secondary complications of type 2 **diabetes**, such as cardiovascular disease. Copyright .COPYRGT. 2002 John Wiley & Sons, Ltd.

L8 ANSWER 27 OF 36 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1993:321644 BIOSIS
DOCUMENT NUMBER: PREV199396029994
TITLE: Complementation analysis of patients with intact peroxisomes and impaired peroxisomal beta-oxidation.
AUTHOR(S): McGuinness, M. C. [Reprint author]; Moser, A. B. [Reprint author]; Poll-The, B. T.; Watkins, P. A. [Reprint author]
CORPORATE SOURCE: Kennedy Krieger Inst., Johns Hopkins Univ. Sch. Med., Baltimore, MD 21205, USA
SOURCE: Biochemical Medicine and Metabolic Biology, (1993) Vol. 49, No. 2, pp. 228-242.
CODEN: BMMBES. ISSN: 0885-4505.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Jul 1993
Last Updated on STN: 31 Aug 1993

AB Complementation analysis, using peroxisomal beta-oxidation of very long chain fatty acids (VLCFA) as the criterion for complementation, is useful

in the study of patients who are suspected of having a single enzyme defect in the peroxisomal beta-oxidation pathway. Laboratory findings for these patients include elevated plasma VLCFA and impaired VLCFA oxidation in fibroblasts. Some of these patients have slightly abnormal **phytanic acid** oxidation in fibroblasts. In addition, elevatd levels of bile acid intermediates have been reported in some cases. Plasmalogen synthesis, pipecolic acid levels, and subcellular distribution of catalase are normal. Using complementation analysis, we show that six patients, who were suspected of having a single enzyme defect in the peroxisomal beta-oxidation pathway, are deficient in peroxisomal bifunctional enzyme (enoyl-CoA hydratase (EC 4.2.1.17)/3-hydroxyacyl-CoA dehydrogenase (EC 1.1.1.35)) activity. This group of six patients, deficient in bifunctional enzyme activity, may be subdivided into two complementation groups. It would appear that patients in each of these two groups are deficient in only one of the bifunctional enzyme activities.

L8 ANSWER 28 OF 36 EMBASE

ACCESSION NUMBER: 2005055864 EMBASE
TITLE: Thiazolidinediones.
AUTHOR: Bloomgarden Z.T.
CORPORATE SOURCE: Dr. Z.T. Bloomgarden, Diabetes Center, Mount Sinai School of Medicine, New York, NY, United States
SOURCE: Diabetes Care, (2005) Vol. 28, No. 2, pp. 488-493. .
Refs: 11
ISSN: 0149-5992 CODEN: DICAD2
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
006 Internal Medicine
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Feb 2005
Last Updated on STN: 18 Feb 2005
DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

L8 ANSWER 29 OF 36 MEDLINE on STN
ACCESSION NUMBER: 1999352945 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10424146
TITLE: A case of motor and sensory polyneuropathy with retinitis pigmentosa and diffuse idiopathic skeletal hyperostosis.
AUTHOR: Osoegawa M; Araki E; Arakawa K; Okayama A; Yamada T; Ohnishi A; Kira J
CORPORATE SOURCE: Department of Neurology, Faculty of Medicine, Kyushu University.
SOURCE: Rinsho shinkeigaku = Clinical neurology, (1999 May) Vol. 39, No. 5, pp. 542-5.
Journal code: 0417466. ISSN: 0009-918X.
PUB. COUNTRY: Japan
DOCUMENT TYPE: (CASE REPORTS)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Japanese
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199909
ENTRY DATE: Entered STN: 12 Oct 1999
Last Updated on STN: 3 Mar 2000
Entered Medline: 24 Sep 1999
AB We here report a 53-year-old man who presented with motor and sensory polyneuropathy, retinitis pigmentosa and diffuse idiopathic skeletal hyperostosis (DISH). He had a 15-year history of **diabetes** mellitus (DM). Visual impairment appeared at 17 years of age. Since age

47, he showed a slowly progressive sensory impairment and muscle weakness of the extremities. On neurological examination, retinitis pigmentosa and severe muscle atrophy, muscle weakness and sensory disturbance of all modalities in the distal portions of all four extremities were observed. Deep tendon reflexes were absent. A plain X-P showed diffuse ossification of the spinal and extraspinal ligaments. The motor nerve conduction velocities were severely reduced and no sensory nerve action potentials were evoked. The CSF examination revealed an increased protein level without pleocytosis. The sural nerve biopsy showed a marked onion bulb formation and a loss of the myelinated nerve fibers, which could not be solely explained by DM. As the **phytanic acids** levels, beta-lipoprotein, lactate and pyruvate in the sera were within the normal ranges, Refsum disease, Bassen-Kornzweig syndrome and mitochondrial diseases were unlikely in this patient. The presence of demyelinating and axonal polyneuropathy in this patient may have been caused by a common metabolic disturbance which produced both retinitis pigmentosa and DISH.

L8 ANSWER 30 OF 36 EMBASE

ACCESSION NUMBER: 2004518203 EMBASE
TITLE: Diet, fatty acids, and regulation of genes important for heart disease.
AUTHOR: Vanden Heuvel J.P.
CORPORATE SOURCE: Dr. J.P. Vanden Heuvel, Department of Veterinary Sciences, Ctr. Molec. Toxicol./Carcinogenesis, Pennsylvania State University, 226 Fenske Laboratory, University Park, PA 16802, United States. jpv2@psu.edu
SOURCE: Current Atherosclerosis Reports, (2004) Vol. 6, No. 6, pp. 432-440. .
Refs: 85
ISSN: 1523-3804 CODEN: CARUCZ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Dec 2004
Last Updated on STN: 28 Dec 2004

AB Diets rich in omega-3 polyunsaturated fatty acids (n-3 PUFAs), such as alpha-linoleic acid, eicosapentaenoic acid, and docosahexaenoic acid, are associated with decreased incidence and severity of coronary heart disease. Similarly, conjugated linoleic acids (CLAs), which are found in meat and dairy products, have beneficial effects against atherosclerosis, **diabetes**, and obesity. The effects of n3-PUFAs and CLAs are in contrast to fatty acids with virtually identical structures, such as linoleic acid and arachidonic acid (ie, n-6 PUFAs). This article discusses the possibility that cognate receptors exist for fatty acids or their metabolites that are able to regulate gene expression and coordinately affect metabolic or signaling pathways associated with coronary heart disease. Three nuclear receptors are emphasized as fatty acid receptors that respond to dietary and endogenous ligands: peroxisome proliferator activated receptors, retinoid X receptors, and liver X receptors. Copyright .COPYRGT. 2004 by Current Science Inc.

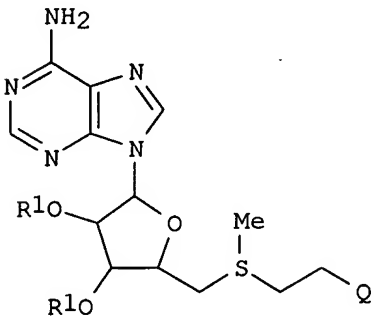
L8 ANSWER 31 OF 36 CAPLUS

ACCESSION NUMBER: 2003:319452 CAPLUS
DOCUMENT NUMBER: 138:314630
TITLE: Orthomolecular sulfo-adenosylmethionine derivatives with antioxidant properties
INVENTOR(S): Wilburn, Michael D.
PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 17 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003078231	A1	20030424	US 2001-886612	20010622

PRIORITY APPLN. INFO.: US 2001-886612 20010622
OTHER SOURCE(S): MARPAT 138:314630
GI



I

AB Disclosed are orthomol. sulfo-adenosylmethionine derivative compds., compns., and their uses for effecting a biol. activity in an animal, such as neurochem. activity; liver biol. activity; heart and artery function; cartilage, bone and joint health; stomach and/or intestinal lining resistance to ulceration; immune function; cell membrane integrity; and pain and inflammation. The compds. of the present invention are further useful for preventing or treating diseases or conditions; treating viral infections, infectious diseases, leukemia, and obesity; and reducing the risk of Sudden Infant Death Syndrome in an animal. The compds. of the present invention are I (R1 = H, C1-C10 alkyl, C2-C10 alkenyl or alkynyl, -C(O)R2; R2 = C1-C10 alkyl, C2-C10 alkenyl or alkynyl; Q = -C(NH3)C(O)AX, -C(COOH)NHX; A = O, N; X = a defined reaction product) or pharmaceutically acceptable salt, ester or solvate thereof. α-(S-adenosylmethionine)-O-tocopherol was prepared from N-Acetyl-S-benzyl-L-homocysteine, α-tocopherol, and 5'-O-p-Tolylsulfonyladenosine.

L8 ANSWER 32 OF 36 EMBASE
ACCESSION NUMBER: 2003409284 EMBASE
TITLE: Reviews: Current topics role of nuclear receptors in the regulation of gene expression by dietary fatty acids (review).
AUTHOR: Khan S.A.; Vanden Heuvel J.P.
CORPORATE SOURCE: J.P. Vanden Heuvel, Department of Veterinary Science, Ctr. Molec. Toxicol./Carcinogenesis, Penn State University, University Park, PA 16802, United States. jpv2@psu.edu
SOURCE: Journal of Nutritional Biochemistry, (1 Oct 2003) Vol. 14, No. 10, pp. 554-567. .
Refs: 142
ISSN: 0955-2863 CODEN: JNBIEL
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Oct 2003
Last Updated on STN: 23 Oct 2003

AB Long chain fatty acids, derived either from endogenous metabolism or by nutritional sources play significant roles in important biological processes of membrane structure, production of biologically active compounds, and participation in cellular signaling processes. Recently, the structure of dietary fatty acids has become an important issue in human health because ingestion of saturated fats (containing triglycerides composed of saturated fatty acids) is considered harmful, while unsaturated fats are viewed as beneficial. It is important to note that the molecular reason for this dichotomy still remains elusive. Since fatty acids are important players in development of pathology of cardiovascular and endocrine system, understanding the key molecular targets of fatty acids, in particular those that discriminate between saturated and unsaturated fats, is much needed. Recently, insights have been gained on several fatty acid-activated nuclear receptors involved in gene expression. In other words, we can now envision long chain fatty acids as regulators of signal transduction processes and gene regulation, which in turn will dictate their roles in health and disease. In this review, we will discuss fatty acid-mediated regulation of nuclear receptors. We will focus on peroxisome proliferators-activated receptors (PPARs), liver X receptors (LXR), retinoid X receptors (RXRs), and Hepatocyte Nuclear Factor alpha (HNF-4α), all of which play pivotal roles in dietary fatty acid-mediated effects. Also, the regulation of gene expression by Conjugated Linoleic Acids (CLA), a family of dienoic fatty acids with a variety of beneficial effects, will be discussed.

L8 ANSWER 33 OF 36 EMBASE
ACCESSION NUMBER: 80174252 EMBASE
DOCUMENT NUMBER: 1980174252
TITLE: [Vegetable oils - analysis and dietary application]. PFLANZLICHE OLE - IHRE ANALYTIK UND DIATETISCHE VERWENDUNG.
AUTHOR: Schilcher H.; Nissler A.
CORPORATE SOURCE: Wissenschaftl. Abt., Johann Georg Fink GmbH & Co., D-7033 Herrenberg, Germany
SOURCE: Physikalische Medizin und Rehabilitation, (1980) Vol. 21, No. 3, pp. 141-156. .
CODEN: PMDRBC
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
019 Rehabilitation and Physical Medicine
LANGUAGE: German
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 9 Dec 1991
Last Updated on STN: 9 Dec 1991

AB Not only former epidemiological investigations but also some new studies have shown how important vegetable oils with a high content of polyunsaturated fatty acids can be in the medical practice. In the following fields of application, suitable vegetable oils are indicated. In case of existing hyperlipidemia (particularly types II and IV depending on nutrition), i.e., if, apart from therapeutic treatment, it is absolutely necessary to influence the serum cholesterol level in a dietary manner. As additional dietary treatment of an existing arteriosclerosis and as preventive modulation of arteriosclerotic risk factors. As additional dietary treatment in case of hypertension and age-induced **diabetes** mellitus. For the replacement of animal fats which, in contrast to vegetable oils, are rich in saturated fatty acids, in case of adiposis (during a reduction diet one has to renounce saturated fatty

acids) and in case of bile and liver diseases as vegetable oils are more easily compatible. Generally, as part of a healthy nutrition because such a diet must contain all the essential nutrients and therefore also polyunsaturated fatty acids sufficiently. Based on several analytic data, one cannot only establish the physiological value of vegetable oils but can also draw conclusions on the manufacturing method and the refining process.

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ACCESSION NUMBER: 2006073515 EMBASE
TITLE: Decoding the pyramid: A systems-biological approach to nutrigenomics.
AUTHOR: Kaput J.
CORPORATE SOURCE: Dr. J. Kaput, Laboratory of High Speed Computing and Informatics, NCMHD Center of Excellence in Nutritional Genomics, University of California, Davis, One Shields Avenue, Davis, CA 95616, United States. jkaput@ucdavis.edu
SOURCE: Annals of the New York Academy of Sciences, (2005) Vol. 1055, pp. 64-79. .
Refs: 44
ISSN: 0077-8923 CODEN: ANYAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 003 Endocrinology
017 Public Health, Social Medicine and Epidemiology
020 Gerontology and Geriatrics
021 Developmental Biology and Teratology
022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2006
Last Updated on STN: 10 Mar 2006

AB Nutritional genomics, or nutrigenomics, seeks to understand the effects of diet on an individual's genes and health. Nutrigenomics is a systems-biological science that can be explained by five principal tenets: (1) improper diets in some individuals and under some conditions are risk factors for chronic diseases; (2) common dietary chemicals alter gene expression and/or genome structure; (3) the influence of diet on health depends upon an individual's genetic makeup; (4) some genes or their normal common variants are regulated by diet, which may play a role in chronic diseases; and (5) dietary interventions based upon knowledge of nutritional requirements, nutritional status, and genotype can be used to develop individualized nutrition plans that optimize health and prevent or mitigate chronic diseases. Optimal nutrition may also influence the aging process. .COPYRGT. 2005 New York Academy of Sciences.

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ACCESSION NUMBER: 2005491091 EMBASE
TITLE: Urological oncology: Prostate cancer.
AUTHOR: Walsh P.C.
SOURCE: Journal of Urology, (2005) Vol. 174, No. 5, pp. 1823-1826. .
ISSN: 0022-5347 CODEN: JOURAA
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 016 Cancer
017 Public Health, Social Medicine and Epidemiology
028 Urology and Nephrology
029 Clinical Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Dec 2005

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DATA NOT AVAILABLE FOR THIS ACCESSION NUMBER

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ACCESSION NUMBER: 79210583 EMBASE
DOCUMENT NUMBER: 1979210583
TITLE: [Course of Refsum's disease treated by diet]. REFSUM KRANKHEIT UND IHR VERLAUF BEI DIATETISCHER BEHANDLUNG DURCH 2.5 JAHRE. KLINIK, BIOCHEMISCHE UND NEUROPATHOLOGISCHE DATEN.
AUTHOR: Lenz H.; Sluga E.; Bernheimer H.; et al.
CORPORATE SOURCE: Neurol. Inst., Univ. Wien, Austria
SOURCE: Nervenarzt, (1979) Vol. 50, No. 1, pp. 52-60. .
CODEN: NERVAF
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
008 Neurology and Neurosurgery
029 Clinical Biochemistry
022 Human Genetics
LANGUAGE: German
SUMMARY LANGUAGE: English

AB A report is given on the first case of Refsum disease observed in Austria. Treatment for it lasted 2 1/2 years. This was dietetic (Steinberg-/Stokke diet, plasmapheresis), which brought improvement of the clinical, biochemical and electrophysiological changes. Comparative bioptic examinations on the sural nerve made it possible to recognize and analyze widespread demyelinations and showed a regression of these and also considerable remyelinations and regenerations after almost 2 years' diet. The difficulties of dietetic therapy are examined in detail, and also its restorative effects on peripheral nerve tissue. There is a discussion on the relationship between the quantity of the biochemical changes and the manifestation of symptom-provoking changes with regard to the myelin.

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